Contractile speed and fatigue of adductor pollicis muscle in multiple sclerosis

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ABSTRACT: The purpose of the study was to investigate differences in contractile speed, force, and fatigability of the adductor pollicis muscle between 12 patients with multiple sclerosis (MS) and 8 sedentary control subjects matched for age and gender. There were no differences between the patients with MS and control subjects with respect to the percentage of maximal muscle force that could be recruited during voluntary effort (95.5 ± 3.9% and 98.2 ± 2.0%, respectively, $P = 0.10$), the stimulation frequency/force and force/velocity relationships, the rates of force development and relaxation, fatigue resistance, and the recovery rate of adductor pollicis muscle. However, previous results from the same group of MS patients showed that quadriceps femoris muscle force and resistance to fatigue were reduced. Therefore, our data support the clinical experience that, in patients with MS, lower limb muscle function is more or earlier affected than upper limb muscle function.
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CONTRACTILE SPEED AND FATIGUE OF ADDUCTOR POLLICIS MUSCLE IN MULTIPLE SCLEROSIS

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Patients with multiple sclerosis (MS) experience symptoms of impaired motor function and muscle weakness. In addition, they often suffer from excessive skeletal muscle fatigue,[21,26,29,30] although excessive muscle fatigue is not always found in patients with MS.[23] Muscle weakness and fatigue in MS patients are probably not caused simply by a reduced capacity for voluntary muscle activation due to demyelination and axonal degeneration in the central nervous system.[20,27] There is also evidence for significant intramuscular causes of weakness and fatigue.[30] For example, a reduced oxidative capacity[17,18] and an increase in the proportion of fast muscle fibers[17] have been found in tibialis anterior muscles of MS patients, perhaps as the result of a reduced physical activity level.[17,24] For clinicians and physical therapists it is important to know the extent to which skeletal muscle properties deteriorate as a result of MS, because weakened skeletal muscles can be trained and such training may help patients to improve their functioning in daily life.

Disuse leads to a decrease in the resistance to fatigue, possible muscle atrophy, and eventually to slow-to-fast fiber type transformations,[13] as reflected by the increased contractile speed that has clearly been demonstrated in paraplegic patients.[11] Although with MS the disuse of skeletal muscle is less extreme than after spinal cord injury, a reduction of the fatigue resistance[21,30] and an increase in the proportion of fast (type II) muscle fibers[17] have been reported for tibialis anterior muscle. However, there are no indications of a disuse-related increase of muscle speed with MS. The speeds of isometric force development and relaxation were found to be unaffected[6] or even decreased in electrically activated quadriceps[27] and tibialis anterior[21,30] muscles. However, these earlier studies were performed on lower limb muscles and only the rates of isometric force

Abbreviations: EDSS, Expanded Disability Status Scale; Fo, maximal isometric force; MS, multiple sclerosis; MVC, maximal voluntary contraction; P_{max}, maximal power output; V_{max}, maximal shortening velocity; V_{opt}, optimal shortening velocity (for maximal power output)
Key words: fatigue; multiple sclerosis; muscle speed; physical therapy; rehabilitation
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development and relaxation were measured. We have therefore studied a hand muscle and also focused on shortening contractions.

Changes in contractile speed of muscle fibers will alter force production during shortening contractions, resulting in a change of the force/velocity relationship.[15] In clinical tests, force (torque) output of a muscle (group) at different imposed movement velocities can be measured and the force/velocity relationship obtained can be used to describe dynamic muscle function. However, even in healthy individuals it is very difficult to obtain valid force/velocity relationships of skeletal muscles in vivo. In addition to the shortening velocity of the contractile elements (muscle fibers), which is the primary interest in the present study, other (confounding) factors, such as the degree of muscle activation, shortening-induced force losses, and effects of series elastic elements (tendon), will determine the in vivo torque production during concentric muscle contractions.[8] In patients with MS, muscle activation is compromised during maximal voluntary isometric contractions,[6] and a pronounced reduction of motor unit firing rate has been found under these circumstances.[27] It is likely that muscle activation becomes even more compromised during maximal voluntary shortening contractions when movement duration and time for muscle activation is limited and action potentials have to be generated over a short time and at a relatively high frequency[5,33] to guarantee a maximal active state of the muscle.

In the present investigation, the different parameters of muscle speed in patients with MS have been studied in the adductor pollicis muscle, which can be maximally activated with electrical nerve stimulation, thereby bypassing the central nervous system. An additional advantage of this muscle is that its temperature is easily controlled. This is important because disuse may lead to a decrease in baseline muscle temperature[12] and even a small reduction in muscle temperature will decrease muscle speed[9,12] and may reduce fatigability.[9] It was hypothesized that in patients with MS there would be a loss of adductor pollicis muscle force, an increase of contractile speed, and a reduction of the resistance to fatigue.

MATERIALS AND METHODS

Subjects. Twelve patients with MS (6 women, 6 men), aged 42.1 ± 7.0 years (mean ± SD; range 26–51 years), participated in the study. Eight nonactive to mildly physically active individuals (4 women, 4 men), with a mean age 48.1 ± 4.3 years (range 43–56 years), served as a non-MS control group. All patients were ambulatory, some with the assistance of a cane. Expanded Disability Status Scale (EDSS)[19] ratings were between 2 and 6 (mean 3.3 ± 1.0), and the time since diagnosis was 8.5 ± 5.1 years. All patients were on interferon-β Ia maintenance treatment, but no other medications. There was no spasticity in the upper limb and, on standard clinical evaluation, upper limb motor function was found to be only mildly impaired: the Scripps motor score for left upper extremity was 4.2 ± 1.0 (range 3–5 on a scale from 1 to 5, with 5 indicating normal function).[32]The study was approved by the local ethics committee and all subjects took part after providing informed consent.

Force Recording and Stimulation. Methods for stimulating the adductor pollicis and force recording are given in detail elsewhere.[7] Briefly, the subject sat in an adjustable chair with the left forearm supinated, and the hand was held horizontally and securely fixed with the thumb abducted and in contact with a vertical pin. The pin was attached to a strain gauge mounted to the support below the plane of the hand. The forces reported in the present study are those applied by the thumb at the vertical pin. When the thumb was fully adducted, its length axis was parallel with the length axis of the index finger and this position was defined as the 0° thumb angle. Because the vertical pin of the force transducer was placed between the thumb and the index finger, the smallest thumb angle at which forces could be measured was 36°. It was possible to increase thumb angle up to 74° (maximal abduction) before anatomical limits were approached. Thus, during shortening contractions, the maximal angular displacement was 38°. Timing and duration of stimulation, onset and speed of motor movement, and data sampling frequency (1000 Hz) of the force and length signal were computer controlled.

The adductor pollicis muscle was activated by percutaneous electrical stimulation of the ulnar nerve at the wrist with constant current unidirectional square-wave pulses of 100-μs duration (Model DS7, Digitimer, Ltd., Welwyn Garden City, UK) at different frequencies. The current was set at 30% above the stimulus that produced maximal isometric tetanic force. Force records of isometric contractions were filtered (fourth-order low-pass digital Butterworth filter, 50-Hz cutoff frequency) and differentiated to obtain the maximal rate of force development and relaxation during each isometric contraction.

To maintain a constant muscle temperature, the subject's hand and forearm were immersed in a water bath at 45.0°C for 20 min prior to the test, and

during the experiment a lamp was used to warm the hand. Skin temperature measured with a thermocouple (diameter 0.25 mm; Thermo Electric International BV, Warmond, The Netherlands) secured with sports tape over the adductor pollicis was between 35.5° and 36.5°C. This procedure will lead to a muscle temperature of approximately 37°C.[9]

Experimental Protocol. All measurements were performed at a thumb angle of 51°, which is optimal for force production, although the angle force relationship is very flat over the range (36°–74°) of applied thumb angles.[7] In all cases there were 2-min rest periods between contractions to prevent fatigue. The first and last contraction were electrically activated (80 Hz, 1-s duration) and the average ($n = 20$) force difference between these contractions was 3.0 ± 2.0%, which confirms that no fatigue occurred during the measurements. For the other measurements, the sequence is explained in what follows.

First, the isometric force/stimulation frequency relationships were obtained using 1-s contractions at different imposed stimulation frequencies (1, 10, 20, 50, 80, and 100 Hz). To obtain the maximal rate of isometric force development a 100-ms contraction at 300 Hz was imposed.

Second, the subject's ability to maximally voluntarily activate the adductor pollicis muscle was tested. A 100-ms pulse train (200 Hz) was imposed on the relaxed muscle at the 51° thumb angle. This was immediately followed by a maximal voluntary contraction (MVC) of the thumb adductors on which the 100-ms, 200-Hz pulse train was superimposed (Fig. 1). Untrained subjects often need several attempts before they are able to produce a maximal contraction. Therefore, the MVC measurement was repeated five times. The attempt with the highest MVC value was used to calculate the difference between the force during MVC and during MVC with the superimposed electrical activation. By comparing the size of the extra electrically evoked force production on top of the MVC with the size of the electrically evoked contraction just before the MVC, we calculated the extent to which the subject was able to voluntarily activate the adductor pollicis muscle (see later also).

Third, a force/velocity curve was constructed using short isovelocity contractions at seven different angular velocities (0°/s, 76°/s, 153°/s, 229°/s, 306°/s, 382°/s, and 458°/s) applied in random order as described in detail elsewhere.[8] It is important, particularly at the highest speeds, that the muscle reaches its maximum active state as fast as possible. To achieve this, muscles were stimulated with relatively high pulse frequencies during these isovelocity shortening contractions at 100 Hz (0°–229°/s) and 300 Hz (306°–458°/s). The thumb was adducted twice at each imposed velocity, once with and once without stimulation of the adductor pollicis (i.e., passive shortening). At each velocity, the passive force was subtracted from the total force trace to provide a measure of the active force. In all cases, the velocity was imposed on the thumb by the motor system and kept constant during thumb adduction (muscle shortening).

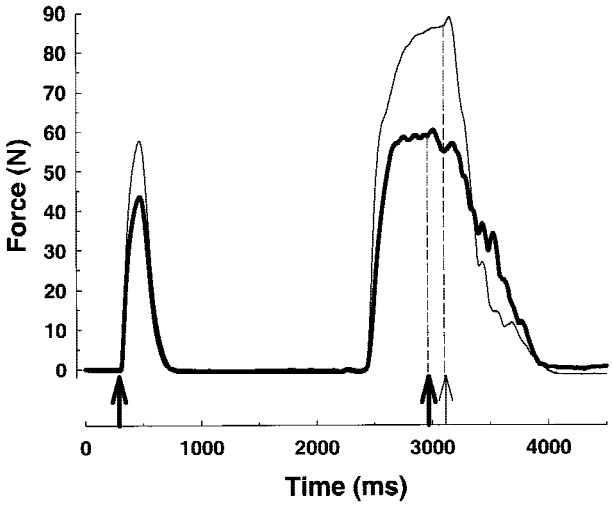

FIGURE 1. Central activation during maximal voluntary thumb adduction. Force records of superimposed electrical activation (train of 100-ms duration and 200-Hz pulse frequency) on the force plateau during a MVC. Superimposition was applied at t = 2935 ms in the control subject (bold arrow) and t = 3110 ms in the patient (thin arrow). The extra force on top of the plateau force during MVC was expressed as a percentage of tetanic force produced by electrical activation of the relaxed muscle (at t = 300 ms). This percentage was subsequently subtracted from 100% to obtain a measure for central activation, which was 96.3% for the patient (thin record) and 97.0% for the control subject (bold record).

Subsequently, the muscle was fatigued with 60 isometric contractions (50-Hz stimulation frequency), each of 500-ms duration, with a 1.50-s rest between contractions (total duration 2 min). To monitor the recovery phase, 500-ms (50-Hz) contractions were applied at 0.75, 1.5, 3, 6, and 12 min following the end of the fatigue run.

Data Analysis. To construct stimulation frequency/isometric force relationships, forces were expressed as a percentage of the maximal isometric force (usually obtained with 80-Hz stimulation).

Force oscillation amplitude was recently introduced as a sensitive measure for the rates of force

rise and relaxation, because force oscillation (ripple) depends on the speed of isometric force development as well as the speed of muscle relaxation.[11] To obtain force oscillation amplitude, the amplitude of three consecutive oscillations in the 10-Hz contractions was divided by the mean force during those oscillations.

The maximal rate of isometric force development during a single contraction is attained following the first (few) stimulation pulses and the rate of force development was expressed as the force change per unit time, expressed as a percentage of the maximal isometric force (% force/ms).[9]

Three different indices for relaxation speed were obtained in the present study: maximal relaxation rate (% force/ms); the time for force to decrease from the highest value following the last stimulus to half that value (early half-relaxation time); and the time needed for force to fall from 50% to 25% (late half-relaxation time).[9] Early half-relaxation time is relatively more sensitive to changes in calcium reuptake by the sarcoplasmic reticulum, whereas late half-relaxation time depends more on changes in crossbridge cycling rate. The relaxation indices were calculated from the 50-Hz tetani.

Extra electrically evoked force production on top of the MVC indicated that the muscle was not maximally activated voluntarily. Central activation was calculated using the following formula[31]: central activation (%) = [1 − (extra force/peak force of the 200-Hz train)] × 100.

Although other formulas have been used to evaluate central activation, it is generally accepted that superimposition of a train of pulses is a reliable method, particularly when the motor nerve is stimulated.[16] In addition, we used the same formula in a recent study on quadriceps femoris muscle function in the same group of patients.[6]

Forces were obtained from the force plateau (see Fig. 2 in De Ruiter et al.[8]) during the isovelocity shortening contractions. From this plateau, force at the 51° thumb angle was used to construct the force/velocity relationship of the muscle. Data points were fitted (least squares) to a hyperbola described by the Hill equation.[14] Optimal shortening velocity (V_{opt}) was defined as the velocity of shortening giving the highest power output (P_{max}) on the velocity power curve, which was derived from the force/velocity curve. Maximal shortening velocity (V_{max}) was determined as the intercept of the Hill curve with the velocity axis. In addition, the curvature index, a/Fo, was calculated.

Isometric force and the rates of force development and relaxation during the fatigue protocol and subsequent recovery phase were expressed as a percentage of the value obtained during the first contraction (= 100%). To obtain a measure for speed of recovery, the differences between the last contraction of the fatigue run and the first measurement (45 s) following the fatigue run were expressed as a percentage of the difference between the first and last contraction of the fatigue run (= 100%).

Statistical Analyses. The results are presented as mean ± SD. A two-factor ("disease" and "gender") analysis of variance, with repeated measures ("time") when appropriate, was used to test for significant differences ($P < 0.05$) between the groups.

RESULTS

Maximal Isometric Force. Maximal stimulated isometric force (100 Hz) was not different ($P = 0.18$) between the patients (60.8 ± 18.4 N) and control subjects (67.8 ± 15.9 N), but in both groups the male subjects were significantly stronger (75.3 ± 15.8 N) than the females (52.0 ± 9.4 N). The gender difference was also seen during maximally voluntary effort, in which males produced 96.1 ± 18.7 N and females 73.4 ± 14.5 N. During MVC, the patients produced significantly ($P = 0.02$) less force (78.1 ± 15.9 N) than the control subjects (94.6 ± 22.5 N). There were, however, no gender or MS-related differences with respect to the percentage of the maximal muscle force that could be recruited during voluntary effort: 95.5 ± 3.9% and 98.2 ± 2.0% for the MS patients and control subjects, respectively ($P = 0.10$; Fig. 1). It should be noted that the higher force values obtained during MVC compared with maximal electrical stimulation were due to the contribution of synergistic muscles during maximal voluntary adduction of the thumb, whereas with electrical stimulation only the adductor pollicis was activated.

Rates of Force Development and Relaxation. Figure 2 shows that, at all frequencies of stimulation, relative force production was very similar ($P > 0.4$) between the groups. A significant difference was not found for any of the parameters between the groups. MS versus control values were, respectively: 1.24 ± 0.13% and 1.22 ± 0.10% force/ms (maximal relaxation rate); 108 ± 9 ms and 109 ± 9 ms (early half-relaxation time); 31 ± 6 ms and 31 ± 5 ms (late half-relaxation time); 1.22 ± 0.12 and 1.21 ± 0.17% force/ms (maximal rate of force development); and 0.44 ± 0.14 and 0.51 ± 0.14 (force oscillation amplitude, $P = 0.29$).

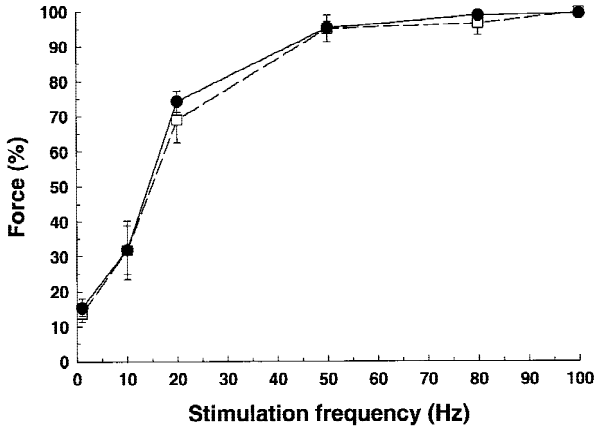

FIGURE 2. Stimulation frequency/isometric force relationship of adductor pollicis muscle in MS. Isometric force production of adductor pollicis muscle of control subjects (filled circles, solid curve) and MS patients (open squares, dashed curve) during 1-s tetanic activation at different stimulation frequencies. Forces (mean ± SD) are expressed relative to the maximal isometric force (=100%) in each subject (obtained at 80 Hz in most subjects). There were no statistical differences between the groups.

Shortening Contractions. During isovelocity shortening contractions there were no differences in relative force production between the groups (Fig. 3). MS versus control values for the calculated parameters were, respectively: 1036 ± 208°/s and 950 ± 227°/s (V_{max}; $P = 0.47$); 270 ± 45°/s and 251 ± 49°/s ($V_{opt;}$ $P = 0.44$); 43.4 ± 17.5 N°/s and 45.7 ± 14.7 N°/s (P_{max}; $P = 0.72$); and 0.15 ± 0.07 and 0.16 ± 0.04 (a/Fo; $P = 0.80$). A gender-related difference was found for maximal power production, which was significantly higher in the men (53.4 ± 16.6 N°/s) than in the women (35.2 ± 9.3 N°/s).

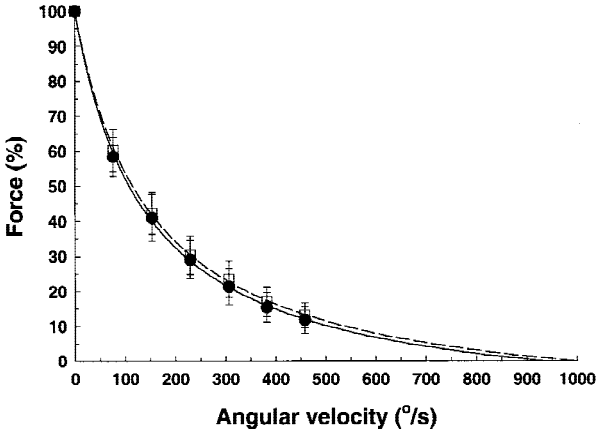

FIGURE 3. Force/velocity relationship of adductor pollicis muscle in MS. Force/velocity relationships of adductor pollicis muscle of control subjects (filled circles, solid curve) and MS patients (open squares, dashed curve). Forces (mean ± SD) are expressed relative to the maximal isometric force (=100%) in each subject. There were no statistical differences between the groups.

Fatigue. Tetanic force, early and late half-relaxation time, maximal rates of force relaxation, and development of the first, 30th, and 60th contraction of the fatigue run and of the contraction at t = 838 s (where the muscle had completely recovered) were not different between the groups (Fig. 4). Although the fatigue-induced changes in tetanic force were not statistically different ($P = 0.23$), from the data presented in Figure 4A it seems there may have been a greater decline of force in the patients at the end of the fatigue run (at t = 120 s). However, even an analysis of variance (ANOVA) for repeated measures using only the data from the 30th (halfway, at t = 60 s) and 60th (last) contraction (at t = 120 s) of the fatigue run did not result in a significant differ-

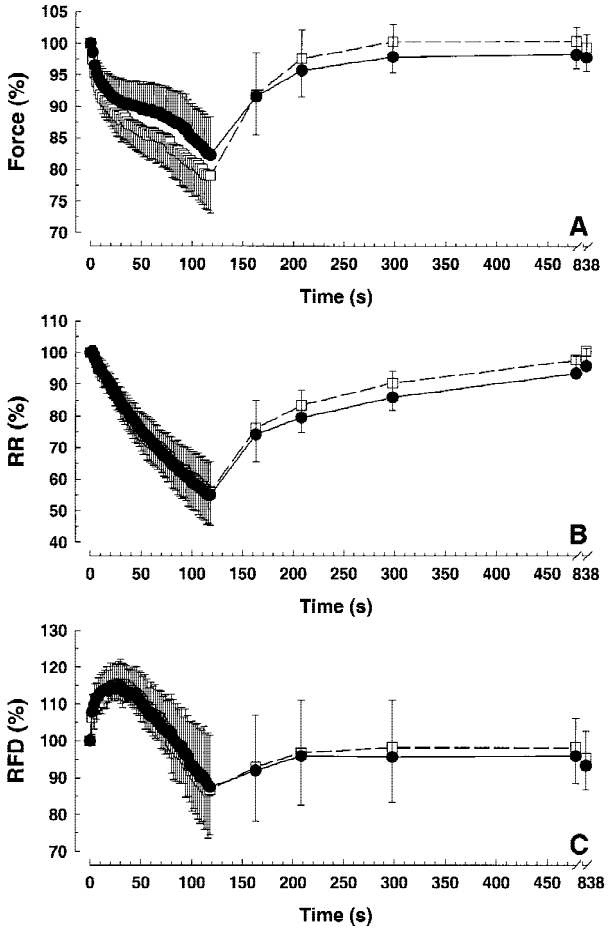

FIGURE 4. Muscle fatigue and recovery of adductor pollicis muscle function. Relative changes of force **(A)**, maximal relaxation rate (RR) **(B)**, and maximal rate of force development (RFD) **(C)** during (0–120 s) and following (120–838 s) repetitive isometric activation for control subjects (filled circles, solid curve) and MS patients (open squares, dashed curve). Values (mean +SD for the control group and −SD for the MS group) are expressed as a percentage of the first contraction (= 100%). There were no statistical differences between the groups.

ence ($P = 0.13$) between the groups. Recovery of tetanic force was also similar between the groups: 45 s following the fatigue run 59.8 ± 22.7% and 55.2 ± 23.4% of the lost force had recovered in the MS and control groups, respectively ($P = 0.67$).

DISCUSSION

In the present study we have investigated whether the rates of force development and relaxation during isometric and force production during isovelocity shortening contractions are affected in patients with MS. We were unable to detect any differences between the patients with MS and a control group in the contractile characteristics of adductor pollicis muscle. In addition to the rates of force development and relaxation, the force/velocity relationship and maximal power production of the electrically activated adductor pollicis muscle were also similar in the patients and control subjects. Moreover, there were no significant differences with respect to adductor pollicis muscle fatigability and recovery rate. Consequently, we had to refute our hypothesis that there would be changes in contractile properties and fatigability of adductor pollicis muscle in patients with MS.

Rates of Force Development and Relaxation. In the present study, a noninvasive method was used to obtain information about the contractile properties of skeletal muscle. The central nervous system was bypassed with electrical nerve stimulation, and force output and parameters of muscle speed and fatigue were measured. An increase of muscle speed may occur with a slow-to-fast fiber-type transformation following disuse. However, adductor pollicis muscle speed was found not to be different in patients with MS compared with the subjects in the control group. A greater proportion of fast fibers in tibialis anterior muscle of MS patients has been reported by others,[17] but in another study by the same group significantly slower (instead of a faster) muscle relaxation was found in the tibialis anterior muscle of patients with MS, and this was attributed to impaired calcium kinetics in the muscle.[30] Moreover, short-term immobilization of the thumb has been shown to lead to a slowing of adductor pollicis muscle relaxation.[10] In the same study, however, a faster rate of isometric force development was found following immobilization. According to Duchateau et al.,[10] the former is not, but the latter is, consistent with a transition of the myosin ATPase from slow to fast. Indeed, changes in the rates of force development and relaxation may not accurately describe changes in shortening speed of the contractile elements. Myosin-specific ATPase activity is the most important determinant of shortening speed of the contractile elements[2]; in addition to the myosin heavy-chain composition, several other factors, such as the speed of calcium handling and the series elasticity of the muscle, make important contributions to the rates of isometric force development and relaxation. In the present study, we did not find any indications for impaired calcium handling; that is, the rates of isometric force development and relaxation, the force oscillation amplitude during 10-Hz stimulation, and the force/frequency relationship were not affected in MS.

Shortening Contractions. To evaluate contractile function under dynamic conditions, force output during concentric contractions over a broad range of imposed velocities was investigated. The results do not indicate that shortening speed of the muscle fibers had changed in the patients' muscle, and values for V_{max} and V_{opt} were similar to those of control subjects. Some studies investigating force output during voluntary concentric muscle contractions have shown relatively greater losses of force at higher shortening velocities in MS patients compared with healthy subjects.[1,3,25] However, particularly in MS patients in whom there is evidence that maximal motor unit firing rates are reduced,[27] it is likely that muscle activation becomes submaximal during voluntary shortening contractions, especially at higher speeds of movement when the time for muscle activation is very limited and action potentials have to be generated at a high frequency.[5,33] In the present study we deliberately bypassed the central nervous system and our approach differed from previous studies on concentric muscle force and MS[1,3,25] in two other aspects. First, we measured force at relatively high speeds of movement and, second, we were able to accurately control adductor pollicis muscle temperature, which is not possible in the larger arm and leg muscles.[28] Temperature control is important because small changes of muscle temperature affect contractile speed (and fatigability).[9] Based on our results we are confident in concluding that, in the present group of MS patients, no changes had occurred in the speed characteristics of the adductor pollicis muscle.

Fatigue. All patients reported that they suffered from some form of "fatigue" in daily life, which were general (central) feelings of fatigue as well as fatigue of certain muscle groups during specific activities, such as climbing stairs. In the present study, there were no differences between the patient and control

groups with respect to fatigability and recovery rates of the different parameters investigated. However, in the same group of patients, quadriceps femoris muscle fatigue was found to be enhanced.[6] In addition, a reduced rate of phosphocreatine resynthesis was reported for the tibialis anterior muscle of MS patients.[18] Muscle fatigue and recovery processes are highly dependent upon the oxidative capacity of the muscle and, therefore, the present findings suggest that oxidative metabolism was unaffected in the adductor pollicis muscle of the patients.

Temperature. We found adductor pollicis muscle function to be unaffected in patients with MS despite the high intramuscular temperature (approximately 37°C).[9] In many patients with MS, symptoms are exacerbated at higher temperatures,[22] a phenomenon attributed to a decreased capacity of demyelinated nerve fibers to conduct action potentials at higher core temperatures.[4] The present findings accord with the notion that the processes affected by an increase of temperature in MS patients are of a central origin, because muscle function was intact even following heating of the arm.

Disuse. Changes in contractile characteristics of muscles in patients with MS may, at least partly, be due to disuse secondary to the disease. However, any reduction in general physical activity level, which has been found with MS,[17,24] does not necessarily imply that the adductor pollicis muscle is also less (intensely) used. We assessed neither the quantity nor the quality of adductor pollicis muscle activity of our patients in their daily life. Therefore, the lack of differences between groups in the present study may be because any disuse was not sufficient to cause significant changes in the contractile characteristics of the adductor pollicis muscle.

Upper versus Lower Limb Muscles and Clinical Implications. Another possible explanation for the lack of differences between the groups in the present study may be that an upper limb muscle was investigated and that upper limb muscle function is less affected in MS than lower limb muscle function.[29] Nevertheless, Schwid et al.[29] found greater fatigue in the elbow extensors and hand-grip muscles in patients with MS. Moreover, Sheean et al.[31] showed that, during a 45-s MVC, adductor pollicis muscle force decreased significantly more in patients with MS than in healthy control subjects. However, in the present investigation, the patients were less disabled (EDSS 3.3 ± 1.0) than in the studies by Schwid et al.[29] (EDSS 5.5 ± 1.3) and Sheean et al.[31] (EDSS 5.4 ± 1.9).

All patients in the present study also participated in a recent study on quadriceps femoris muscle function and MS.[6] In that study, both voluntary and electrically induced forces were lower and muscle fatigability was higher in patients than in the control subjects. Moreover, some of the present findings suggest that adductor pollicis muscle function in our patients was in the early stages of decline. For example, the forces generated by the patients during maximal voluntary thumb adduction were significantly lower than those of the control subjects, whereas central activation (>95%) was not statistically different ($P = 0.10$) between the groups. This suggests that, in the patients, the thumb adductors as a group were intrinsically weaker, possibly due to atrophy of the muscle fibers. This suggestion was supported by the tendency for a lower maximal tetanic force of the adductor pollicis muscle in the patients. However, it cannot be excluded that the force exerted by the synergists was more affected by MS than adductor pollicis muscle force. This would explain why we did find a significant decrease of maximal voluntary force but not of stimulated contractions. With more statistical power (more subjects) more concrete conclusions may have been reached. Nevertheless, in combination with our recent findings,[6] the present data seem to support the idea that lower limb muscle function is more or earlier affected in MS than upper limb muscle function. This is an important finding, because, at an early stage of the disease when muscle function has not yet been greatly affected, a limited amount of additional muscle training may be sufficient to prevent, or at least to slow, further deterioration of muscle function.

In conclusion, the present study did not find differences in contractile speed, force, and fatigability of electrically activated adductor pollicis muscle between patients with MS and a control group. Moreover, based on contractile characteristics, there were no indications for impaired calcium handling. Furthermore, muscle speed, fatigability, and recovery following fatigue were not affected in the patients' muscle. These findings suggest that there were no changes with respect to fiber type composition and oxidative capacity of the muscle. The present findings are partly in contrast to the results of a recent study in which the same patients participated and which showed that quadriceps muscle strength and fatigue resistance were affected. Our data support the clinical experience that, in patients with MS, lower limb muscle function is more or earlier af-

fected than upper limb muscle function. This is an important finding not only because of its implications for clinical testing of muscle function with MS, but also with regard to the development of rehabilitation programs.

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